In contrast, a lower mortality rate was associated with cardiac surgery for the other shock pts (38% vs 71% p < 0.0001).

In conclusion, VSR/MR caused shock in 14% of pts registered with shock and AMI. VSR/MR pts were less likely to have anterior MI and more likely to have posterior MI, with shock onset at a significantly longer time post MI. Pts. with mechanical complication (VSR/MR) have the worst prognosis of all shock pts due to AMI. To impact on the very high mortality once shock has occurred, early detection and treatment of VSR/MR are required prior to shock onset.

1061-98 Characteristics and Outcome of Patients with Cardiogenic Shock Due to Right Ventricular Dysfunction: A Report from the SHOCK Trial Registry

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To evaluate the clinical characteristics and outcome of pts with acute myocardial infarction (AMI) and cardiogenic shock (CS) due to right ventricular (RV) dysfunction, we queried the SHOCK trial registry, a prospectively collected database which includes all pts with suspected CS due to AMI not enrolled in the randomized SHOCK trial. RV CS was defined on clinical grounds. Twenty-one pts with predominant RV CS were compared with 105 pts with predominant LV CS (CI ≤ 2.2 L/min). Clinical characteristics are shown:

	Predominant RV	Predominant LV	p-value
Age (yrs)	65.9 ± 10.1	69.3 ± 9.8	0.16
Male (%)	57.1	53.3	0.81
Anterior MI (%)	15	56.4	0.001
Prior MI (%)	20.0	39.8	0.13
PCWP (mmHg)	18 ± 5	25 ± 9	0.009
C.I. (L/min)	1.7 ± 0.4	1.6 ± 0.4	0.65
Lytic Rate (%)	42.9	43.8	1.00
IAB (%)	38.1	64.8	0.03

In-hospital mortality was high in both groups (RV 47.6%, LV 62.9%, p = 0.23). Interestingly, pts with RV CS were treated with PTCA less frequently than pts with LV CS (19.1 vs 41.9%, p = 0.053). Therefore, despite having fewer anterior MIs, a trend towards fewer prior MIs and lower filling pressures than pts with LV CS, pts with RV CS also have a high in-hospital mortality. The infrequent use of PTCA in this group may be an important observation and should be the subject of future study.

1061-99 New Device Intervention in Cardiogenic Shock

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Few data are available regarding percutaneous transluminal coronary revascularization (PTCR) in patients with cardiogenic shock (CS). The efficacy and feasibility of new device (ND) [atherectomy, laser, and stent] intervention in this critically ill population has not been explored. We reviewed our experience of PTCR in CS patients, and identified 53 patients (age 68 ± 12 years, 29 [55%] men), treated in our laboratory from January 1994 through April 1996. CS was defined as 2 or more of the following: clinical hypoperfusion, dependence on vasopressor support, SBP < 90 mmHg, PCWP ≥ 18 mmHg, CI ≤ 2.1 L/min/BSA.

Clinical, angiographic and outcome variables were collected via a retrospective chart review. Sixteen (30%) patients died despite interventional therapy. Therapy was categorized as balloon angioplasty alone (POBA) in 31; or new device (ND) in 21. One patient who died after his lesion could not be crossed was excluded from the analysis of device use and mortality.

Mortality was significantly lower (p = 0.03) in patients with ND (9.5%) compared to POBA (41.9%). An analysis of stent vs. no stent revealed a significant difference in mortality, 6.3% vs. 37.8% (p = 0.04), in favor of stent use. Thirty-nine (75%) patients had CS associated with acute infarction (AMI), 12 others had unstable angina. All deaths occurred in the 39 patients who presented with AMI. In this subgroup, both ND and stent use were associated with a lower mortality (p = 0.08/0.08).

Mortality (All patients)	Mortality (AMI patients)
ND: 2/21 (9.5%) p = 0.03	ND: 2/13 (15.4%) p = 0.08
POBA: 13/31 (41.9%)	POBA: 13/26 (50%)
Stent: 1/15 (6.3%) p = 0.04	Stent: 1/10 (10%) p = 0.08
No stent: 14/37 (37.8%)	No stent: 14/29 (48.3%)

Conclusion: ND can be safely used in CS patients. Coronary stenting may be associated with a lower mortality.

1061-100 Endotoxemia Alters Splanchnic Capacitance

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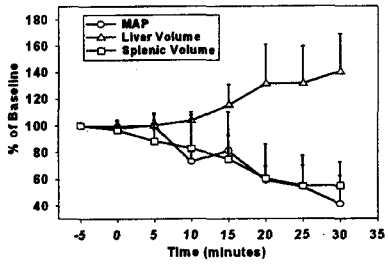
It is known that unresuscitated endotoxemic (LPS) shock reduces cardiac output. Since the splanchnic circulation constitutes a major portion of the total capacitance vasculature, it is possible that an increase in splanchnic blood volume leads to a decrease in venous return and subsequently cardiac output. However, it is unknown if LPS affects splanchnic capacitance vessels. We assessed the effects of Ecoli LPS (10 μg/kg over 5 minutes) on individual (liver, splenic, mesenteric) and total splanchnic blood volume in 5 unresuscitated farm swine. Splanchnic blood volume was measured using Tc^{99m} labeled erythrocytes and radionuclide imaging. Mean arterial pressure (MAP) and splanchnic volumes were recorded every 5 minutes during a basal period and after the start of LPS infusion (0 min).



The figure shows MAP, liver and splenic volume as a percent of baseline before (−5 to 0 min) and after LPS (5–30 min). LPS decreased splenic volume by 45% and MAP by 60% while liver volume increased by 40% (p < 0.01). Mesenteric and total splanchnic volume were unchanged. It is likely that the lack of change in total splanchnic volume was due to the counteracting affects of changes in splenic and hepatic volume. Since total splanchnic volume was unchanged, the reduction in cardiac output in unresuscitated LPS shock is not due to pooling of blood volume in the splanchnic capacitance circulation.

1061-101 Multiple Triggering Mechanisms of Vasovagal Syncope: A Patient-Controlled Study

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Recent studies suggest multiple reflexogenic mechanisms may trigger a vasovagal response in different patient populations. We hypothesized that the triggering mechanisms may vary even in the same patients. We examined hemodynamic changes of 6 patients (F/M = 4/2; mean age = 45 yr) who had vasovagal syncope induced by both 45 min head up tilt alone and tilt + Iso infusion (0.05 μg/kg/min). The protocol order was randomized. Impedance cardiography and plethysmography was used to collect data. Hemodynamics during both protocols (Tilt ± Iso) were compared in each patient. Mean responses while supine, and at peak response just before onset of symptoms (reflecting triggering mechanisms of incipient syncope) are shown:

Hemodynamics	Mean Change, Tilt:					
	Supine			Peak Response before Sx.		
	Iso−	Iso+	p	Iso−	Iso+	p
HR, bpm	63	96	< 0.0005	29%	84%	< 0.05
SBP, mmHg	138	131	n.s.	1%	−12%	n.s.
DBP, mmHg	77	65	< 0.05	0 %	−13%	n.s.
CO, L/min	6.3	10.8	< 0.05	−11%	103%	< 0.05
SV, cc	103	114	n.s.	−32%	5%	< 0.05
EDV, cc	178	206	n.s.	−15%	3%	< 0.005
TPR, dyn-s/cm^3	1286	796	< 0.005	13%	−54%	< 0.005

Observations: 1) While supine, Iso increased HR and CO, and decreased DBP and TPR. 2) During tilt alone, EDV decreased (reflecting preload), and TPR increased (afterload) prior to onset of symptoms. 3) Addition of Iso to tilt significantly increased HR, SV, and CO (cardiomotor tone) and reversed tilt-induced changes in EDV and TPR.

Conclusions: 1) Triggering mechanisms of vasovagal syncope can differ in the same patient. 2) These observations may explain why response to therapy targeting triggering mechanisms may be less than satisfactory.